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Acute Kidney Injury

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Continuing Education Activity

Acute kidney injury (AKI), previously known as acute renal failure, denotes a sudden and often reversible reduction in kidney function, as measured by increased creatinine or decreased urine volume. AKI can be differentiated into prerenal, intrarenal, and postrenal etiologies, and these etiologies can be overlapping and interrelated. Distinguishing the causes of AKI is fundamental to effectively and efficiently treating AKI, which improves patient outcomes. This activity reviews the evaluation and management of acute kidney injury and highlights the role of the interprofessional team in managing patients affected by this condition.

Objectives:

- Identify the risk factors and clinical indicators associated with acute kidney injury to facilitate early recognition and intervention.
- Differentiate between prerenal, intrinsic renal, and postrenal causes of acute kidney injury, utilizing diagnostic tools and clinical assessment to guide appropriate treatment strategies.
- Implement evidence-based guidelines for preventing and managing acute kidney injury, including optimizing fluid resuscitation, adjusting medications, and addressing underlying causes.
- Communicate effectively with patients and their families, providing clear explanations of acute kidney injury, its causes, and the proposed treatment plan.

Access free multiple choice questions on this topic.

Introduction

Acute kidney injury (AKI), previously called acute renal failure (ARF), denotes a sudden and often reversible reduction in kidney function, as measured by glomerular filtration rate (GFR).[1][2][3] However, immediately after a renal insult, blood urea nitrogen (BUN) or creatinine (Cr) levels may be within the normal range, and the only sign of AKI may be a decline in urine output. AKI can lead to the accumulation of water, sodium, and other metabolic products. AKI can also result in other electrolyte disturbances. AKI is a prevalent condition, especially among hospitalized patients, and can be seen in up to 7% of hospital admissions and 30% of ICU admissions. Several criteria have been used to identify AKI, such as RIFLE, AKIN (Acute Kidney Injury Network), and KDIGO (Kidney Disease: Improving Global Outcomes) criteria. Among these, KDIGO is the most recent and most commonly used tool. According to KDIGO, AKI is the presence of any of the following:[4]

1. Increase in serum creatinine by 0.3 mg/dL or more (26.5 µmol/L or more) within 48 hours
2. Increase in serum creatinine to 1.5 times or more than the baseline of the prior 7 days
3. Urine volume less than 0.5 mL/kg/h for at least 6 hours

The RIFLE criteria define 3 categories of impairment—risk, injury, and failure—and 2 categories of long-term renal outcomes—loss and end-stage renal disease (ESRD). Whichever criterion shows the most impairment is used for classification. When baseline Cr is unknown, a baseline GFR between 75 and 100 mL/min is assumed, or the Modification of Diet in Renal Disease (MDRD) equation can be used to calculate an estimated baseline Cr.[4][5]

1. Risk: Cr ↑ of 1.5x baseline, GFR ↓ of 25%, or urine output (u/o) <0.5mL/kg/h for 6 h
2. Injury: Cr ↑ of 2x baseline, GFR ↓ of 50%, or urine output (u/o) <0.5mL/kg/h for 12 h
3. Failure: Cr ↑ of 3x baseline, GFR ↓ of 75 %, Cr ≥4.0, or urine output (u/o) <0.5mL/kg/h for 12 h
4. Loss: Loss of kidney function for over 4 weeks
5. ESRD: Loss of kidney function for over 3 months

The AKIN criteria are based on the RIFLE criteria and are also called the "modified RIFLE" criteria. While the RIFLE and KDIGO systems have higher sensitivity than AKIN, all 3 have similar predictive abilities for in-hospital mortality.[4]

Etiology

The impetus for glomerular filtration is the pressure difference between the glomerulus and Bowman's space. This pressure gradient is affected by the renal blood flow and is under the direct control of the combined resistances of afferent and efferent vascular pathways. For most causes of AKI, renal blood flow reduction is a common pathologic pathway for declining GFR. The pathophysiology of AKI has traditionally been divided into three categories: prerenal, intrinsic renal (ie, intrarenal), and postrenal. Each of these categories has many different associated causes, and some causative factors of AKI have overlapping mechanisms of injury.[6][7]

The prerenal form of AKI is due to any cause of reduced blood flow to the kidney. This may be part of systemic hypoperfusion resulting from hypovolemia or due to selective hypoperfusion of the kidneys, such as resulting from renal artery stenosis or aortic dissection. However, tubular and glomerular function tends to be initially normal. A few examples of prerenal AKI mechanisms are listed below:

1. Hypovolemia: hemorrhage, severe burns, and gastrointestinal fluid losses such as diarrhea, vomiting, and high ostomy output.
2. Hypotension from decreased cardiac output: cardiogenic shock, massive pulmonary embolism, acute coronary syndrome.
3. Hypotension from systemic vasodilation: septic shock, anaphylaxis, anesthesia administration.
4. Renal vasoconstriction: NSAIDs, iodinated contrast, amphotericin B, calcineurin inhibitors, hepatorenal syndrome.
5. Glomerular efferent arteriolar vasodilation (causing intraglomerular hypotension): ACE inhibitors, angiotensin receptor blockers.

Intrinsic renal causes include conditions that affect the glomerulus or tubule, such as acute tubular necrosis and acute interstitial nephritis. This underlying glomerular or tubular injury is associated with the release of vasoconstrictors

from the renal efferent pathways. Prolonged renal ischemia, sepsis, and nephrotoxins are the most common causes. It is worth mentioning that prerenal injury can convert into a renal injury if the offending factor's exposure is prolonged enough to cause cellular damage. A few examples of this mechanism are listed below:

1. Acute tubular necrosis(ATN): ischemia from prolonged prerenal injury; drugs such as aminoglycosides, vancomycin, amphotericin B, and pentamidine; iodinated contrast; rhabdomyolysis; intravascular hemolysis
2. Acute interstitial nephritis (AIN): Drugs such as beta-lactam antibiotics, penicillins, NSAIDs, proton pump inhibitors (PPIs), and 5-ASA; infection; autoimmune conditions (systemic lupus erythematosus (SLE), IgG-related disease); and hereditary AIN.
3. Glomerulonephritis: anti-glomerular basement membrane disease, immune complex-mediated diseases (such as SLE, post-infectious glomerulonephritis, cryoglobulinemia, IgA nephropathy, IgA vasculitis).
4. Intratubular obstruction: monoclonal gammopathy (such as in multiple myeloma), tumor lysis syndrome, hemolytic anemia, and toxins such as ethylene glycol.

Postrenal etiology for AKI includes obstructive causes, which lead to congestion and urinary backflow of the filtration system, leading to a shift in the filtration driving forces. A noteworthy fact is that a unilateral obstruction may not always present as AKI, especially if the obstruction is gradual, because a normal working contralateral kidney may compensate for the function of the affected kidney. Pathological disturbances can occur within 2 hours of obstruction, starting with decreased filtration at the level of the glomerulus due to increased upper urinary tract pressure. This results in decreased renal perfusion, inflammation, tubular atrophy, and interstitial fibrosis. Eventually, bladder atony, periglomerular fibrosis, chronic interstitial nephritis, and secondary FSGS can develop. Weeks or months of obstruction can lead to ESRD. Once obstruction is released, post-obstructive diuresis occurs in up to 50% of patients and should be monitored for severe complications of hypovolemia and electrolyte abnormalities. The most common etiology of postrenal AKI is bladder outlet obstruction, which is often due to prostatic hypertrophy in older men, pelvic masses in older women, and nephrolithiasis in younger patients.[8]

1. Renal/ureteral calculi can present in the renal calyces, renal pelvis, bladder, or urethra. Size and location are the determining factors of AKI, and this is a significant etiology in those with a solitary kidney. Struvite and cystine stones grow especially rapidly and commonly cause obstruction.
2. Tumors, blood clots, and neurogenic bladder cause mechanical ureteral outlet obstruction. Blood clots can be a result of bladder or urinary tract malignancy.
3. Urethral obstruction is the most common cause of prostate enlargement in older men. The obstruction can also be caused by retroperitoneal fibrosis, pregnancy, fecal impaction, pelvic organ prolapse, pelvic masses/malignancy, or phimosis.

Epidemiology

AKI is commonly seen in hospitalized patients. In the United States, 1% of all hospital admissions have AKI on admission. During hospitalization, the approximate incidence rate of acute kidney injury is 2% to 5%, and it occurs in up to 67% of patients admitted to the intensive care unit. AKI is thus an important contributor to more extended hospital stays and patient morbidity.[9][10][11]

Pathophysiology

The pathogenesis of AKI is etiology-driven. The common endpoint in acute tubular necrosis is a cellular insult secondary to ischemia or direct toxins, which results in effacement of the brush border, cell death, and decreased function of tubular cells. One intrarenal cause is intratubular obstruction—such as by pigments such as myoglobin,

crystals such as uric acid in tumor lysis syndrome, or immunoglobulin light chains, as seen in monoclonal gammopathy—which can also lead to the same result. Other intrarenal mechanisms of injury are glomerulonephritis or acute interstitial nephritis, which can be due to immune-mediated injury of the vasculature, inflammatory responses, and immune complex deposition leading to glomerular and tubular damage. Postrenal pathophysiology is usually related to urinary reflux, causing decreased renal perfusion, tubular atrophy, and interstitial inflammation.

Histopathology

Histopathology can reliably differentiate the intrinsic renal pattern of AKI from others; however, histopathology may not be reliable at narrowing down a specific cause in every situation. Renal biopsy is an invasive procedure and is usually only pursued in cases where a significant impact on management is expected, such as suspected glomerulonephritis or if multiple etiologies are possible. Immunofluorescence patterns, electron microscopy, and staining for fibrosis and inflammatory cells can help differentiate various causes in such cases.

History and Physical

The history and physical exam should focus on determining the etiology of AKI and the progression timeline. If the history points towards hypovolemia or hypotension, then the treatment is guided towards volume repletion. Providers should look for inciting events such as diarrhea, nausea, vomiting, which may have caused volume loss, or any over-the-counter drugs such as NSAIDs. Differentiating between AKI and chronic kidney disease (CKD) is essential, as CKD itself is not an uncommon risk factor for AKI. A history suggestive of CKD can include symptoms such as chronic fatigue, anorexia, nocturia, disturbed sleep-wake cycle, polyuria, and pruritis. Moreover, a careful review of past medical history may reveal comorbid conditions that can help narrow down the etiology of AKI, such as cirrhosis or a history of blood clots requiring anticoagulation. History and physical examination are essential in AKI because labs often cannot provide a clear answer as to the etiology of AKI.

The most common causes of AKI in hospitalized patients are as follows:

1. ATN, 45%
2. Prerenal disease, 21%
3. AKI superimposed on CKD, 13%
4. Urinary tract obstruction, 10%
5. Glomerulonephritis or vasculitis, 4%
6. AIN, 2%
7. Atheroemboli, 1%

A history of urine output may give clues as to the cause of AKI. The following are some associations:

1. Oliguria favors AKI.
2. Sudden anuria suggests acute urinary tract obstruction, acute glomerulonephritis, or vascular blockage.
3. Polyuria suggests either a concentrating tubular defect, such as acute interstitial nephritis, or postobstructive nephropathy.

A detailed physical exam can provide extremely valuable information in establishing the etiology of AKI. A crucial part of the physical exam should be orthostatic vital signs, which may signify hypovolemia. Several organ systems can provide clues regarding the cause of AKI. Some of them are:

1. Skin: livedo reticularis, digital ischemia, butterfly rash, and purpuras to suggest vasculitis. A maculopapular rash may indicate drug-induced AIN. Needle marks may suggest IV drug use, which can cause endocarditis. Livedo reticularis and skin infarcts are also seen with cholesterol emboli disease. Decreased skin turgor suggests hypovolemia.
2. Eyes and ears: jaundice is present in liver disease, band keratopathy is present in multiple myeloma, diabetes mellitus can show microscopic retinopathy, and blood vessel narrowing is associated with hypertension. Keratitis, iritis, and uveitis may be present in autoimmune vasculitis. Hearing loss is associated with Alport disease.
3. Cardiovascular system: pulse rate, blood pressure, and jugulovenous pulse can indicate volume status. Irregular rhythm may indicate electrolyte imbalance-related arrhythmias. Pericardial friction rub may be heard in uremic pericarditis, and a heart murmur is often heard in the case of endocarditis. Embolic disease can present as cyanotic extremities.

Evaluation

Evaluation of AKI should include a thorough search for all possible etiologies of AKI, including prerenal, intrarenal, and postrenal disease. Noting the time of onset of AKI can be useful when dealing with hospitalized patients. For example, if a sudden creatinine rise is noted, an inciting factor usually occurs in the 24 to 48 hours preceding this onset. It is imperative to look for any radiologic studies involving the use of iodinated contrast agents, a common cause of AKI. It is also important to review the medications the patient is receiving and determine if the doses need to be modified. ACE inhibitors and ARBs are often the co-contributors to AKI.

All patients presenting with AKI warrant a comprehensive metabolic panel. Urine electrolytes can also help suggest an etiology of the AKI. Urine studies should be checked for electrolytes, protein, osmolality, and albumin-to-creatinine ratios. Older patients may warrant serum and urine protein electrophoresis (SPEP and UPEP) to rule out monoclonal gammopathy and multiple myeloma. Renal ultrasound can be helpful if obstructive causes are suspected. CT scans are another important radiographic modality and can be used to look for nephrolithiasis or urolithiasis. Urine microscopy can also provide important clues about the etiology, such as muddy brown casts seen in ATN or white blood cell casts sometimes seen in AIN. Sterile pyuria is the most specific sign of acute interstitial nephritis.[12][13]

Although creatinine is the most commonly used serum chemistry to evaluate for AKI, there is evidence that other biomarkers may be more sensitive and may be elevated earlier in the course of AKI compared to creatinine. Some of these include neutrophil gelatinase–associated lipocalin (NGAL), which can be measured in the plasma and urine, kidney injury binding protein-1 (KIM-1), retinol-binding protein, cystatin C, α/β microglobulin, and urine uromodulin. The use of these biomarkers is not widely available and has not yet been validated in wide-scale studies.[14][15]

A kidney biopsy is an excellent but infrequently utilized tool. It is usually indicated in patients with rapidly declining renal function without apparent cause or when multiple etiologies of AKI are possible. It is a test with some associated risks, such as bleeding, and should be used cautiously if a solitary kidney or coagulopathy is present.

There are markers of tubular function that can be calculated to help distinguish between prerenal, intrarenal, and postrenal causes, such as the fractional excretion of sodium and urea and urine osmolality; however, these parameters are affected by many drugs commonly used in clinical practice such as diuretics and are not always diagnostic. No single marker can be reliably used in isolation to distinguish among the causes of AKI, and clinical presentation must be considered.

Treatment / Management

Many cases overlap between prerenal and ATN types of AKI.[16][17] The best way to determine if the AKI is prerenal or not is a fluid challenge. If there is no contraindication, all patients with acute renal dysfunction should receive a fluid challenge. This requires close monitoring of urine output and renal function. If the renal function improves with fluid, this indicates prerenal AKI. Acute tubular necrosis and other intrarenal causes are often slow to recover and can take weeks to months for complete recovery of renal function. Diuretics may be required during the oliguric phase of ATN if significant volume overload develops. It is also important to avoid further kidney insults, such as nephrotoxic drugs. In addition, many medications must be renally adjusted once a patient develops AKI. Dietary ingestion of potassium and phosphorus should also be monitored.

If hyperkalemia develops, it needs to be managed expeditiously. Approaches to lower potassium in the body include:

1. Dietary restriction
2. Insulin, IV dextrose, and beta-agonists
3. Potassium-binding resins
4. Calcium gluconate to stabilize the cardiac membrane if EKG changes are present
5. Dialysis for nonresponsive hyperkalemia

Some AKI patients tend to develop volume overload, which should be corrected as early as possible to avoid pulmonary and cardiac complications. Euvolemic state can be achieved with the help of diuretics, which is a cornerstone in managing such patients. Usually, high doses of IV furosemide are needed to correct volume overload in AKI patients; however, it plays no role in converting oliguric AKI to non-oliguric AKI.

In some cases, short-term renal replacement therapy is needed for AKI until the kidney function recovers. Some indications for RRT are severe and nonresponsive hyperkalemia, uremic pericarditis, and pulmonary edema. This is seen especially in the oliguric phase of acute tubular necrosis, where the patient is prone to develop multiple electrolyte and acid-base abnormalities as well as fluid overload.[18] Dialysis in this setting is usually performed through a temporary venous catheter when required. Continuous renal replacement therapy can also be utilized in patients who cannot tolerate hemodialysis due to hypotension. It is a much slower, continuous type of dialysis. Correction of some of the metabolic abnormalities, along with dialysis, may be required. Metabolic acidosis is one such instance where systemic administration of citrate or bicarbonate is often required to maintain a suitable blood pH. The requirement for renal replacement therapy should be reevaluated daily. Renal replacement therapy is usually required for short periods, ranging from a few days to a few weeks; however, some cases can take months to recover and may require intermittent RRT support.

Other treatments are directed at the etiology of the AKI. Examples include administering vasoactive medications and colloids for the treatment of hepatorenal syndrome, cautious diuresis in cardiorenal syndrome, immunosuppressive medication for various glomerulonephritides or vasculitides, or steroids for AIN. Postrenal obstruction may need to be relieved operatively in certain situations. For example, benign prostatic hypertrophy may require surgical intervention, and obstructive calculi may require stenting and lithotripsy.

Differential Diagnosis

Differential diagnoses to be considered in AKI include renal calculi, acute or chronic kidney disease, hypovolemia, gastrointestinal bleeding, decreased cardiac output, urinary tract infection, and urinary obstruction. Rarely, ingesting excessive protein or dietary supplements can cause elevated creatinine levels unrelated to kidney disease.[19]

Prognosis

Most prerenal AKI cases recover completely with correction of the underlying insult if treated early; however, the persistence of the underlying insult may lead to ATN, in which case the damage may not be completely reversible. Another consideration is that although recovery from individual episodes may be complete or partial, repeated AKI can lead to a cumulative worsening of renal function. Therefore, it is essential to monitor these patients closely to normalize renal function or until a new baseline is established. The in-hospital mortality rate for AKI is 40% to 50%, and the mortality for ICU patients is more than 50%. Other prognostic factors include:

1. Older age
2. Duration of illness
3. Fluid balance
4. Diuretic use
5. Oliguria
6. Hypotension
7. Inotropic support
8. Multiorgan involvement
9. Sepsis
10. Number of transfusions

Over the long term, at least 12% to 15% of patients with AKI may require permanent dialysis. Mortality is increased in patients with high APACHE III scores, advanced age, and persistent creatinine elevation.[20][21]

Complications

Several complications may associate AKI with mortality. Some of these complications are directly associated with AKI and can easily be gauged (hyperkalemia, volume overload, metabolic acidosis, hyponatremia); however, the effect of other complications on AKI-related mortality, such as inflammation and infection, is more difficult to assess. The most common complications include metabolic derangements such as:

1. Hyperkalemia can lead to arrhythmias and, if severe, is considered a medical emergency.
2. Metabolic acidosis may necessitate systemic administration of bicarbonate or citrate buffers.
3. Hyperphosphatemia can usually be prevented by decreasing dietary ingestion or using phosphate binders.
4. Other adverse effects include pulmonary edema from volume overload and peripheral edema from an inability to excrete body water. This is especially common in the oliguric phase of ATN. It may necessitate the use of diuretics or renal replacement therapy.

The other organ-related complications include:

1. Cardiovascular: Heart failure secondary to fluid overload is attributable to oliguric AKI. Arrhythmias can be secondary to acidosis and electrolyte abnormalities. Cardiac arrest can result from metabolic derangements. Myocardial infarction and pericarditis are also rare complications.
2. Gastrointestinal (GI): Nausea, vomiting, GI bleeding, and anorexia can be associated with AKI. Amylase may be elevated with AKI, so the measurement of serum lipase should be used to diagnose pancreatitis if clinical suspicion is present.

3. Neurologic: CNS-related signs of uremia include lethargy, somnolence, disturbed sleep-wake cycle, and cognitive impairment.

Deterrence and Patient Education

For patients who have developed AKI, several factors should be advised to preserve renal function, such as avoiding nephrotoxic agents and dehydration. NSAIDs are also known to cause interstitial nephritis, which can lead to the development of AKI or the worsening of existing AKI. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are known to affect renal autoregulation, and the use of these medications in AKI depends on the clinical picture.

Enhancing Healthcare Team Outcomes

AKI has significant morbidity and mortality if left untreated. All healthcare workers should be educated on the condition, its causes, and potential complications. At the first signs of creatinine elevation, pharmacists should ensure that the patient is not on nephrotoxic medications. The patient should be educated that the regular consumption of NSAIDs is harmful to the kidney and that noncompliance with blood pressure medications can worsen kidney injury.

Close follow-up with a nephrologist is highly recommended in all cases of AKI. The patient should have a dietary consult because salt and fluid restriction are vital when managing AKI. Similarly, the patient should avoid a high-potassium diet when there is renal dysfunction. Because AKI induces a catabolic state, the patient should be encouraged to consume adequate protein.[22][23][24]

Primary care providers should be regularly updated about hospitalized patients and upon discharge, especially if patients have ongoing serum chemistry abnormalities. Only through an interprofessional approach can the morbidity of AKI be lowered.

Review Questions

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